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The Genesis 12–19 (G1219) Study: A Twin and Sibling Study of Gene–Environment Interplay and Adolescent Development in the UK

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The Genesis 12–19 (G1219) Study is an ongoing longitudinal study of a sample of UK twin pairs, non-twin sibling pairs, and their parents. G1219 was initially designed to examine the role of gene–environment interplay in adolescent depression. However, since then data have continued to be collected from both parents and their offspring into young adulthood. This has allowed for longitudinal analyses of depression and has enabled researchers to investigate multiple phenotypes and to ask questions about intermediate mechanisms. The study has primarily focused on emotional development, particularly depression and anxiety, which have been assessed at multiple levels of analysis (symptoms, cognitions, and relevant environmental experiences). G1219 has also included assessment of a broader range of psychological phenotypes ranging from antisocial behaviors and substance use to sleep difficulties, in addition to multiple aspects of the environment. DNA has also been collected. The first wave of data collection began in the year 1999 and the fifth wave of data collection will be complete before the end of 2012. In this article, we describe the sample, data collection, and measures used. We also summarize some of the key findings to date.

■ **Keywords:** anxiety, depression, externalizing, longitudinal, sleep, twins

Adolescence is a unique developmental period, characterized not only by biological change but by social upheaval and exposure to a range of novel environmental stressors. Unsurprisingly, behavioral and emotional problems often emerge during this period. In attempting to understand the development of such problems, it is necessary to examine risk factors from multiple domains. For example, it is known that genetic factors play a role in the development of emotional and behavioral problems. However, gene sequence alone cannot explain all of the variance in such problems — environmental risk factors such as maltreatment, deprivation, and stressful life events often play an important role as well. Any understanding of emotional and behavioral problems would also be incomplete without incorporating cognition — psychopathology is often characterized by difficulties in information processing or biases in perception, and elucidating the role of such faulty cognitions in the development of emotional problems is likely to prove vital to our understanding of the etiology of psychological phenotypes.

Because there are so many risk factors related to emotional difficulties in adolescence, and because they span multiple domains (genetic, environmental, cognitive), any attempt to understand the development of emotional problems must consider multiple risk factors together. For example, in recent years it has become apparent that genes and environment correlate and interact with one another (collectively referred to as gene–environment interplay). That is, a person genetically predisposed toward behavioral difficulties is often raised in an environment containing environmental risks that also predispose toward behavioral difficulties (gene–environment correlation). And some-

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times the effects of environmental risk factors may be mediated by genotype, such that people with a certain genotype are more/less sensitive to environmental risk factors than are those with a different genotype (gene–environment interaction). Clearly, genetic and environmental risk factors need to be considered in tandem if researchers are to gain a clear understanding of the etiology of emotional difficulties. If we are to understand the link between gene–environment interplay and phenotype, then it is also important that potential intermediate phenotypes that may lie on the 'pathway' between genes/environment and phenotype are investigated. Given their association with emotional and behavioral problems, cognitive biases may play a mediatory role and provide a link from ultimate causes to phenotypic outcome.

The Genesis 12–19 (G1219) Study was set up to investigate the development of emotional and behavioral difficulties with a specific focus on the interplay between genes, environment, and cognition. G1219 is a nation-wide study of adolescent twins and siblings, aged 12–19 years at the outset, growing up in the United Kingdom. Through the use of structural equation models, twin samples can be used to investigate the relative role of genes and environment in explaining population variance in phenotypes of interest. Because G1219 includes sibling pairs as well as twins, it also has increased power to detect environmental effects common to family members. The inclusion of siblings also means that findings from G1219 may be more generalizable than those using other twin samples. G1219 was established to assess the role of gene–environment interplay in adolescent depression. Once underway, the breadth of the study increased to include a range of emotional and behavioral phenotypes, and associated cognitive factors and environmental experiences.

Depression was selected as the initial target of G1219, owing to evidence emerging prior to the start of the study that in addition to the well-documented increase in prevalence of depression during adolescence (Hankin et al., 1998), there was an associated increase in genetic influence on depression symptoms in this age range (Eley & Stevenson, 1999; Thapar & McGuffin, 1994). Given that new environmental challenges are also known to emerge in adolescence, we proposed that this would also be a good developmental stage at which to explore gene–environment interaction and correlation in adolescent depression. In order to examine these processes, we not only obtained repeated ratings of depression, but also assessments of multiple aspects of the adolescent and their environment. These ranged from measures of the social environment (including parent–child relationships, peer relationships, and romantic relationships) to measures of the home environment and both family-wide and adolescent-specific life events. We were also particularly interested in the question of genetic overlap between depression and anxiety (Eley & Stevenson, 1999) and whether this might reflect cognitive biases associated with each symptom type. In order to explore this further, we included measures of cognitions associated with both anxiety and depression. As the study progressed, further areas of interest developed. First, we became interested in exploring specificity of associations between adolescent depression and different aspects of antisocial behavior (e.g., rule-breaking, aggressive, and oppositional subtypes). Second, we undertook twin analyses of sleep disturbances in young adults, including their association with both emotional and behavioral difficulties. Third, we conducted the first ever twin study of driving behaviors during adolescence and young adulthood.

As well as twin-reported measures, G1219 includes parent-report measures of parent and child phenotypes. The use of multiple raters potentially reduces the impact of reporter bias, thus improving the validity of measured phenotypes. DNA has also been collected from a large proportion of participants. As such, the G1219 study is well placed to examine adolescent emotional and behavioral phenotypes using both quantitative and molecular genetic approaches.

Sample and Data Collection

In the G1219 sample sibling pairs and twin pairs were recruited using two distinct strategies: Sibling pairs originated from the GENESiS study (Sham et al., 2000). The GENESiS sample comprised approximately 40,000 adults, of whom approximately 9,000 had, prior to the start of G1219, indicated that they had children living with them. These 9,000 were sent a letter inviting those with children aged 12–19 years old to take part in G1219. A total of 1,818 adolescent offspring from 1,294 families responded to the G1219 invitation, of whom 1,747 adolescents from 1,241 families were aged 12–19 years (mean 15.01, $SD = 2.09$). This sample included 445 sibling pairs. Siblings had a mean average age difference of 28 months (maximum age difference was 70 months). In order to be able to use the data from these sibling pairs in quantitative genetic analyses, a companion sample of twins was recruited in collaboration with the UK Office of National Statistics. A random selection of the parents of live twins born between 1985 and 1988 were contacted via health authorities and general practitioners. The study contacted 2,947 families, of whom 1,381 participated (1,381 twin pairs from as many families).

Ethical approval for waves 1 –3 of the G1219 was granted by the Research Ethics Committee of the Institute of Psychiatry and the South London and Maudsley NHS trust. Ethical approval for waves 4 and 5 was granted by the Research Ethics Committee of Goldsmiths, University of London.

Informed consent was obtained from all participants aged 16 years and over, and from the parents of participants aged less than 16 years.

The composition of the G1219 sample is illustrated in Figure 1. The number of participants and response rates at each wave are given in Table 1 and elaborated upon below.

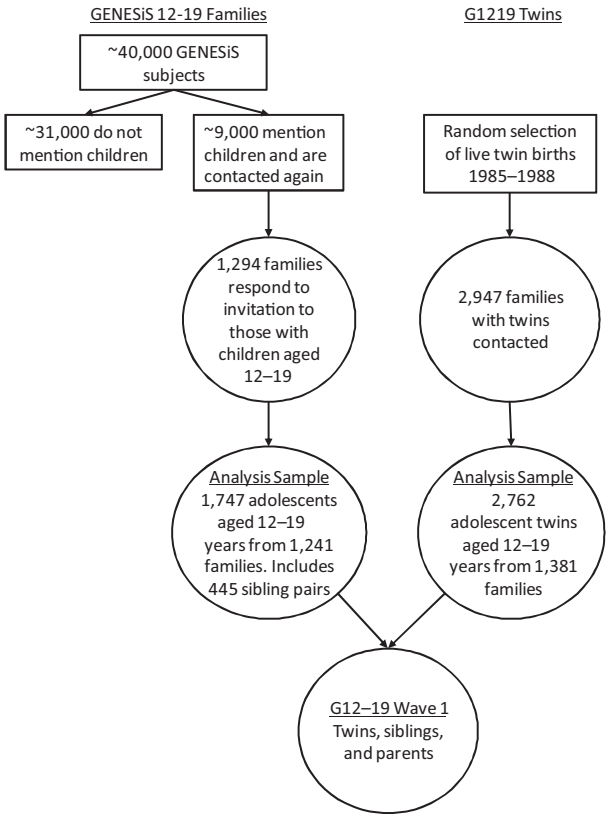

FIGURE 1
Sample recruitment in the G1219 Study.

Wave 1

Data collection for the G1219 study began in the year 1999. Wave 1 took place in three stages: GENESiS families (contacted during 1999–2000), twins born in 1985 (contacted during 2000), and twins born in 1986–1988 (contacted during 2001). In the first wave of data collection, participants were 3,640 adolescents from 1,820 families. The sample of adolescents had an age range of 12–19 years, with a mean age of 14 years. The sample was 52% female. The sample composition is described in Table 1. Short self-report questionnaires were mailed to adolescents and their parents (followed up by two postal reminders). Questionnaires assessed demographics, social problems, and the emotional wellbeing of parents and children. Full details on the measures included at each wave are given in Table 2.

Wave 2

The second wave of data collection focused exclusively on twins and siblings (parents were not surveyed), and the entire sample was contacted at the same time, during 2001–2002. At the second wave, 73% of the wave 1 sample took part, a total of 2,651 participants from 1,372 families. Wave 2 questionnaires were completed an average of 8 months (range = 0.8–22 months) after initial (wave 1) contact. The sample was 56% female with an age range of 12–21 years, and a mean age of 15 years. The sample composition is given in Table 1. For those participants who were among the last to return wave 1 questionnaires, there was only a short gap before wave 2. While this leads to a broad range in terms of time lapsed between waves, for 75% of the sample the time lapsed between waves 1 and 2 was between 4 and 10 months, and was greater than 3 months for 95% of the sample.

At wave 2, questionnaires were posted to participants (followed by two reminders), and included measures assessing (among others) anxiety, anxiety sensitivity, depression, antisocial behavior, attributional style, peer characteristics, parental discipline, life events, and pubertal development (see Table 2).

Wave 3

The third wave of data collection spanned 2003–2004. Questionnaires were posted to twins/siblings and their parents. Twins and siblings reported on their own emotions, behavior, and relationships. Parents completed two questionnaires — one assessing their own emotions and problems, and the other assessing their perceptions of the behavior, emotions, and experiences of their children. At the third wave, data was collected on 1,778 adolescents from 913 families (67% of wave 2 sample; 49% of the wave 1 sample). The full sample composition is given in Table 1. Wave 3 questionnaires were returned on average 25 months (range = 7–41 months) after wave 2, with 75% of the sample returning wave 3 questionnaires 20–31 months after wave 2. The sample was 60% female with an age range of 14–23 years, and a mean age of 17 years. Measures at wave 3 assessed many of the same phenotypes as measured at waves 1 and 2, but also included hyperactivity, substance use, hopelessness, and driving attitudes and behaviors (see Table 2).

Wave 4

The fourth wave of data collection took place in 2007. Twins and siblings who had taken part in waves 2 and/or 3 were traced. This resulted in 2,550 twins/siblings being identified and sent self-report questionnaires in the post (parents were not included in wave 4). Three further postal reminders were sent, along with emails and phone calls. A total of 1,556 individuals from 896 families took part in the study (61% of those targeted and 88% of those participating at wave 3). The sample was 61% female with an age range of 18–27 years, and a mean average age of 20 years. The sample composition is given in Table 1. The measures included in wave 4 overlapped with those from previous waves but also included measures of sleep-related phenotypes, as well as assessments of the personal relationships that participants were engaged in.

TABLE 1
Number of G1219 Pairs (Complete Pairs) Providing Data at Each Wave

	Mean age	Monozygotic pairs		Dizygotic pairs			Sibling pairs			
	(Range)	*M–M*	*F–F*	*M–M*	*F–F*	*OS*	*M–M*	*F–F*	*OS*	Unknown zygosity
Wave 1	14	168	199	138	190	463	109	132	186	235
	(12–19)	(168)	(199)	(138)	(190)	(463)	(109)	(132)	(186)	(235)
Wave 2	15	156	194	124	189	334	69	111	150	45
	(12–21)	(153)	(192)	(122)	(187)	(323)	(54)	(91)	(118)	(39)
Wave 3	17	97	148	71	141	243	39	68	91	15
	(14–23)	(96)	(145)	(68)	(135)	(230)	(35)	(61)	(81)	(14)
Wave 4	20	75	155	76	138	232	44	68	89	19
	(18–27)	(65)	(125)	(53)	(111)	(163)	(28)	(44)	(56)	(15)

Note: M–M = male-male pairs; F–F = female-female pairs; OS = opposite sex pairs.

Wave 5

At the time of writing (July, 2012) data collection for wave 5 of G1219 is underway. The majority of participants are now in their twenties. Measures at wave 5 comprise many of those included in previous waves, as well as additional measures of personality and temperament, sleep-related phenotypes, caffeine intake, as well as other novel measures (e.g., related to coordination; mindfulness). Wave 5 measures are included in Table 1.

DNA

DNA from buccal swabs has been collected by post from a total of 1,237 individuals. Initially DNA was collected at wave 1 from those with very high and very low depression scores in order to explore gene–environment interaction in adolescent depression ($N = 290$ individuals; 75% of those targeted; see Eley et al., 2004a). A small number of samples were collected at wave 3 ($N = 26$ pairs, collected to determine zygosity) with the remaining samples collected following wave 4 ($N = 895$; 61% of those targeted).

Determination of Zygosity

Zygosity was established using a questionnaire measure completed by mothers at waves 2 and 3, assessing physical similarity between twins (Cohen et al., 1975). When zygosity was only available for one or other wave, this rating was used. Where there was disagreement between zygosity ratings at the two waves, buccal cell DNA was obtained (at wave 3; $N = 26$ pairs) and used in making final classifications.

Representativeness of the Sample and Attrition

The representativeness of the G1219 families has been assessed by comparison of wave 1 demographic variables to those detailed in a large survey carried out on a nationally representative sample of parents in Great Britain in 1999 (Meltzer et al., 2000). Levels of parental education were somewhat higher in the G1219 sample, with 39% educated to A-level or above compared to 32% in the nationally representative sample. (In the United Kingdom, A-levels are the highest qualifications that can be obtained while at school, typically achieved by remaining in non-compulsory full-time education until the age of 18.) Parents from the G1219 sample were also more likely to own their own homes (82% compared to 68%).

At each wave of G1219 some participants have dropped out, either because they did not wish to complete further lengthy booklets, because researchers were unable to locate them, or because they had died. At wave 2, attrition was predicted by parental education, housing tenure, and child sex (girls being more likely than boys to remain in the study). At wave 3, attrition was predicted by parental education, housing tenure, delinquency (averaged across siblings), and child sex.

Summary of Key Findings

At the time of writing, 28 empirical papers have been published using data from the G1219 study, with an additional one in press, and three under review (not including the present article). Our key findings to date are summarized below and relate to the following phenotypes: depression, anxiety, antisocial behavior, and sleep disturbances.

Depression

As described above, G1219 was established to examine gene–environment interaction and correlation in adolescent depression. The first paper from the study used only the adolescent offspring of GENESiS participants to explore interactions between serotonergic genes and family environment on adolescent depression. The main finding was that females with two copies of the short allele of the serotonin transporter gene (5HTTLPR), who also came from families subject to elevated environmental risk (low SES, high social problems, adverse life events), were at greater risk of elevated depression (evidence of gene–environment interaction; Eley et al., 2004a; recipient of the Lilly Molecular Psychiatry Award). In our second paper, also using the offspring of the GENESiS sample only, we showed that adolescents in families where parents (and siblings) had

TABLE 2

Measures Included at Each Wave of the G1219 Study

Measures	Parent self-report	Adolescent self-report	Parent report on adolescent
Demographics			
Age and sex	3	1, 3, 4, 5	3
Ethnicity	1, 3		
Education	1, 3	3, 4, 5	
Living arrangements	1, 3	3, 4, 5	
Employment	1, 3	3, 4, 5	
Adjustment and personality			
Depression (short MFQ; Angold et al., 1995)	3	1, 2, 3, 4, 5	3
Anxiety: Spence Child Anxiety Scale (Spence, 1997)		2, 3	
Revised Child Anxiety and Depression Scale (Chorpita et al., 2000)		4, 5	
State-Trait Anxiety Inventory (Spielberger, 1983)		3, 4	
Child Anxiety Sensitivity Index (Silverman et al., 1991)		2, 3	
Adult Anxiety Sensitivity Index (Reiss et al., 1986)		4, 5	
Neuroticism (EPI; Eysenck & Eysenck, 1964)	1		
Antisocial behavior (Achenbach, 1991a; 1991b; Achenbach & Rescorla, 2003)	3	2, 3, 4, 5	3
Hyperactivity (Achenbach, 1991)			3
Callous-unemotional traits (Essau et al., 2006)		4	
Hopelessness (Beck et al., 1974)		3	
Attributional style (Thompson et al., 1998)	3	2, 3	
Prosocial behavior (SDQ; Goodman, 1997)		2, 3	3
Shame (adapted from Andrews et al., 2002)		2	
5-Facet Mindfulness Questionnaire (adapted from Baer et al., 2006)		5	
10-Item Personality Inventory (Gosling et al., 2003)		5	
Affective Reactivity Index (Stringaris et al., 2012a)		5	
Coordination (Elisabeth Hill)		5	
Substance use			
Smoking (Curry et al., 2001)		3, 4, 5	
Alcohol use (Curry et al., 2001)		3, 4, 5	
Use of illicit drugs (Curry et al., 2001)		3	
Caffeine intake (adapted from Kendler & Prescott, 1999)		5	
Health and physical development			
Height and weight (and BMI)		2, 4, 5	
Pubertal development (Petersen et al., 1988)		2	
Driving			
License (Richard Rowe)		3, 4, 5	
Distance driven per week (Richard Rowe)		3, 4, 5	
Attitudes to driving (West & Hall, 1997)		3, 4, 5	
Driving behavior (Reason et al., 1990)		4, 5	
Sleep			
Pittsburgh Sleep Quality Index (Buysse et al., 1989)		4, 5	
Pittsburgh Sleep Quality Index Addendum (Germain et al., 2005)		5	
Insomnia Symptom Questionnaire (Okun et al., 2009)		4, 5	
Morningness/eveningness (Horne and Östberg, 1976)		4, 5	
Sleep quality compared to sibling (Alice Gregory)		5	
Bedroom/bed partners (Alice Gregory)		5	
Epworth Sleepiness Scale (Johns, 1991)		5	
Sleep paralysis (Chris French)		5	
Pre-Sleep Arousal Scale (Nicassio et al., 1985)		5	
Dysfunctional beliefs/attitudes about sleep (Espie et al., 2000)		5	
Experiences			
Social Problems Questionnaire (Corney & Clare, 1985)	1, 3		
Threatening experiences (Brugha & Cragg, 1990)	1, 3	4	
Life events (Coddington & Humphrey, 1984)		2, 3, 4, 5	3
Peers and peer relationships			
Friends: Number		4	
Friends: How many shared with twin/sibling?		2	
Peer deviancy items (Fergusson et al., 2003)		3, 4	
Best friend: Shared with twin/sibling?		2, 4	
Best friend: Friendship quality (Parker and Asher, 1993)		2	
Best friend: Friendship quality (Furman & Buhrmester, 1985)		4	
Characteristics of Friendship Group (adapted from the SIDE; Daniels & Plomin, 1985)		2	
Relationship with parents			
Mother–child and father–child discipline (Dunn et al., 1998, from Hetherington & Clingempeel, 1992)		2	
Romantic relationships			
Demographics of relationship and any children		4, 5	
Dyadic adjustment scale (Spanier, 1976)		4, 5	
Partner delinquency (Fergusson et al., 2003)		4, 5	
Zygosity			
Physical similarity (Cohen et al., 1975)			2, 3
DNA		1, 3, 4*	

Note: *DNA was collected on a subsample of participants at wave 1. At wave 3, DNA was collected for a select few for whom wave 1 and wave 2 zygosity reports were inconsistent. DNA was collected from the majority of the remaining sample at wave 4.

high levels of depression/anxiety and no educational qualifications were at substantially increased risk of elevated depression (a familial risk–environment interaction; Eley et al., 2004b). Continuing with this theme, a subsequent paper took a quantitative genetic approach and, using the entire G1219 sample, showed that the heritability of depression increased in those reporting greater numbers of negative life events or levels of maternal punitive discipline (gene–environment interaction; Lau & Eley, 2008b).

We have also explored gene–environment correlation and the role of cognitive biases in depressive symptoms. For example, we showed that genetic influences on adolescent depression overlapped considerably with those on parental punitive discipline, but also on negative attributional style — a cognitive bias associated with depression (Lau et al., 2006). Adolescent depression has been measured at every wave of G1219, enabling exploration of change and stability in etiological factors associated with depression over time. Lau and Eley (2006) showed that genetic factors at wave 1 contributed to depression at waves 2 and 3. However, there was also evidence for genetic innovation, with new genetic factors contributing to depression at later waves. Environmental effects were specific to each time point. When we explored the longitudinal association between attributional style and depression, we found that genetic factors largely accounted for correlations between the variables both within and across time (Lau & Eley, 2008a).

Anxiety

Gene–environment interaction has also been explored with respect to anxiety, where we found that elevated levels of negative life events were associated with increased genetic influences on panic (Lau et al., 2007). Stressful life events have also been found to play a role in the development of anxiety sensitivity across waves 2, 3, and 4 — predicting concurrent anxiety sensitivity as well as change in anxiety sensitivity across time (Zavos et al., 2012b). Anxiety sensitivity is a marker of cognitive vulnerability to anxiety (Weems et al., 2002). A considerable focus of the project has been to explore the association between anxiety and depression, and the role of both attributional style (traditionally associated with depression) and anxiety sensitivity (traditionally associated with anxiety). We have shown that anxiety sensitivity is as strongly associated with depression as it is with anxiety, and that the structure of the genetic associations largely reflects these phenotypic patterns both in concurrent (Zavos, 2010) and longitudinal (Zavos et al., 2012a) analyses. Of note, as with depression, continuity over time in anxiety sensitivity was also found to be largely due to heritable factors (Zavos et al., 2012a). Most recently, we have begun to explore the structure of anxiety sensitivity (Brown et al., 2012). Our findings indicate that it comprises three dimensions depicting physical, social, and mental anxiety-related concerns. Genetic analyses revealed that the etiology of anxiety sensitivity was best described by a common pathway model, with a single moderately heritable latent 'anxiety sensitivity' factor and three less heritable dimensions representing physical, social, and mental concerns.

Antisocial Behavior

Antisocial behavior is a heterogeneous concept, comprising subtypes with distinct etiologies and developmental pathways (Burt & Neiderhiser, 2009). Most of the G1219 papers focusing on antisocial behavior have distinguished between three subtypes of antisocial behavior: oppositionality (behavior that disregards, or conflicts with, authority), non-violent delinquency, and physical aggression. The first study to do so explored the association between antisocial behavior and depression in wave 2, demonstrating that depressed mood was associated with oppositionality and non-violent delinquency but not physical aggression (Rowe et al., 2006). Negative life events were strongly implicated in the association between delinquency and depressed mood, whereas depressogenic attributional style was implicated in the associations of both delinquency and oppositionality and depressed mood. This paper was followed up by a multivariate genetic analysis of associations between antisocial behavior and depressed mood in wave 2, showing that the correlations between antisocial subtypes and depression were primarily accounted for by genetic overlap (Rowe et al., 2008). Subsequent analyses have shown that the etiological overlap between substance use and antisocial behavior in wave 3 was similar across subtypes of antisocial behavior, although genetic correlations between antisocial subtypes and cannabis use were stronger than those with alcohol consumption (McAdams et al., 2012). Gene–environment interplay has also been investigated in relation to antisocial behavior in G1219 (Button et al., 2008). Findings revealed that antisocial behavior shared genetic overlap with negative life events, maternal punitive discipline, and paternal punitive discipline. When controlling for this overlap, gene–environment interaction was evident such that genetic variance decreased as a function of maternal punitive discipline, but increased as a function of paternal punitive discipline. Most recently, we have explored associations that irritability has with depression and delinquency (Stringaris et al., 2012b). Irritability is of current interest not only as one of two components of Oppositional Defiant Disorder (the other being hurtful and headstrong behaviors; Stringaris & Goodman, 2009), but also because it is a potential variant to depressed mood when diagnosing depression in pediatric samples. Interestingly, in both phenotypic and genetic analyses we found significantly stronger associations between irritability and depression than between irritability and delinquency. Conversely, we found significantly stronger phenotypic and genetic correlations between headstrong/hurtful behaviors and delin-

quency than between headstrong/hurtful behaviors and depression.

Sleep

The first G1219 paper on sleep demonstrated a phenotypic association between poor sleep quality and an 'eveningness preference' (as opposed to a preference for mornings) that was largely accounted for by heritable factors (Barclay et al., 2010). Furthermore, there was substantial genetic overlap between sleep quality and diurnal preference, suggesting that largely the same genes are implicated in both phenotypes. Subsequent papers have demonstrated moderate phenotypic and genetic associations between these two variables and externalizing behaviors (Barclay et al., 2011a), and between sleep quality and internalizing difficulties (Gregory et al., 2011). Evidence for gene–environment correlation has also been found such that genetic factors associated with sleep quality are also involved in the experience of negative life events (Barclay et al., 2011b). In addition, molecular genetic analyses have demonstrated that individuals carrying two copies of the long allele of the 5HTTLPR gene experience significantly poorer sleep quality than those carrying at least one short allele (Barclay et al., 2011b). This is perhaps contrary to what one would expect, given that the short allele typically confers greater risk for psychopathological difficulties in much of the psychopathology literature.

Final Summary and Future Plans

In summary, G1219 has contributed to our understanding of multiple adolescent phenotypes and their interrelationships. Repeatedly, it has been demonstrated that genetic and environmental factors correlate and interact with one another in such a way that their effects cannot truly be understood independently. For example, the genotypes of individuals have been shown to correlate with parental punitive discipline (Button et al., 2008), negative life events (Barclay et al., 2011c), and family environmental risk (Eley et al., 2004b). We have also shown that genetic associations often mirror phenotypic associations, with this extending beyond traditional ratings of emotional and behavioral symptoms to associated cognitive styles. The G1219 sample is now in early adulthood and we continue to follow their development in an effort to better understand gene–environment interplay in behavior and psychopathology, as well as the role of cognitive biases across the lifespan. In the future, we intend to examine the relationship between epigenetic change (specifically DNA methylation) and the development of psychopathology across adolescence. We also hope to collect data on the children of G1219 twins, thus enabling us to examine mechanisms of transmission from one generation to the next.

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